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"Modus Tollens" Probabilized: Deductive and Inductive Methods in Medical Diagnosis

Il "Modus Tollens" Probabilistico: Metodo Deduttivo e Induttivo nella Diagnosi Medica

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Background Medical diagnosis has been traditionally recognized as a privileged field of application for so called probabilistic induction. Consequently, the Bayesian theorem, which mathematically formalizes this form of inference, has been seen as the most adequate tool for quantifying the uncertainty surrounding the diagnosis by providing probabilities of different diagnostic hypotheses, given symptomatic or laboratory data. On the other side, it has also been remarked that differential diagnosis rather works by exclusion, e.g. by *modus tollens*, i.e. deductively. By drawing on a case history, this paper aims at clarifying some points on the issue. Namely: 1) Medical diagnosis does not represent, strictly speaking, a form of induction, but a type, of what in Peircean terms should be called 'abduction' (identifying a case as the token of a specific type); 2) in performing the single diagnostic steps, however, different inferential methods are used for both inductive and deductive nature: modus tollens, hypothetical-deductive method, abduction; 3) Bayes' theorem is a probabilized form of abduction which uses mathematics in order to justify the degree of confidence which can be entertained on a hypothesis given the available evidence; 4) although theoretically irreconcilable, in practice, both the hypothetical-deductive method and the Bayesian one, are used in the same diagnosis with no serious compromise for its correctness; 5) Medical diagnosis, especially differential diagnosis, also uses a kind of "probabilistic *modus tollens*", in that, signs (symptoms or laboratory data) are taken as strong evidence for a given hypothesis *not* to be true: the focus is not on hypothesis confirmation, but instead on its refutation [$Pr(\neg H/E_1, E_2, \ldots, E_n)$]. Especially at the beginning of a complicated case, odds are between the hypothesis that is potentially being excluded and a vague "other". This procedure has the advantage of providing a clue of what evidence to look for and to eventually reduce the set of candidate hypotheses if conclusive negative evidence is found. 6) Bayes' theorem in the hypothesis-confirmation form can more faithfully, although idealistically, represent the medical diagnosis when the diagnostic itinerary has come to a reduced set of plausible hypotheses after a process of progressive elimination of candidate hypotheses; 7) Bayes' theorem is however indispensable in the case of litigation in order to assess doctor's responsibility for medical error by taking into account the weight of the evidence at his disposal.

Index Terms Clinical Methodology, Bayes' theorem

Premessa *La diagnosi medica è stata identificata come un privilegiato campo d'applicazione della cosiddetta "induzione probabilistica". Di conseguenza il teorema di Bayes, che formalizza matematicamente questa forma di inferenza è stato visto come lo strumento più adeguato per quantificare l'incertezza della diagnosi fornendo la probabilità associata alle diverse ipotesi diagnostiche, sulla base dei dati a disposizione (sintomatici o di laboratorio). D'altro canto è stato fatto notare che la diagnosi differenziale lavora piuttosto per esclusione, ad esempio utilizzando il modus tollens, quindi deduttivamente. Utilizzando una case history, il presente articolo mira a chiarificare alcuni punti in questione. Soprattutto: 1) la diagnosi medica non rappresenta, strettamente parlando, una forma di induzione, ma piuttosto ciò che in termini peirceani dovrebbe essere chiamata "abduzione" (che consiste, fra l'altro, nel classificare un caso come token di un type specifico); 2) nell'eseguire i singoli passi diagnostici, vengono utilizzati diversi metodi inferenziali sia di natura deduttiva che induttiva: modus tollens, metodo ipotetico-deduttivo, abduzione; 3) Il teorema di Bayes è una forma probabilizzata di abduzione che utilizza strumenti matematici per legittimare il grado di credibilità dell'ipotesi diagnostica in relazione all'evidenza disponibile; 4) sebbene teoreticamente non conciliabili, in pratica, il metodo ipotetico-deduttivo e bayesiano sono utilizzati nella stessa diagnosi senza grave pregiudizio per la correttezza della soluzione; 5) la diagnosi medica, specialmente la diagnosi differenziale, utilizza anche una sorta di modus tollens probabilistico in quanto i segni (sintomi o dati di laboratorio) vengono presi come evidenza in sostegno della negazione dell'ipotesi: il focus non è tanto sulla conferma quanto sulla confutazione dell'ipotesti [$Pr(\neg H/E_1, E_2, \ldots, E_n)$]. Questo vale specialmente all'inizio di un caso complesso, dove la "scommessa" è tra l'ipotesi che va potenzialmente esclusa e una alternativa indefinita. Questa procedura ha il vantaggio di fornire degli indizi in relazione a quale tipo di informazione cercare e di ridurre il numero delle ipotesi candidate se nessun evidenza conclusiva viene trovata; 6) il teorema di Bayes quale strumento di conferma dell'ipotesi può rappresentare più fedelmente la diagnosi medica, sebbene comunque idealisticamente, quando questa è giunta ad insieme ridotto di ipotesi plausibili alla fine di un processo di progressiva eliminazione di tutte le ipotesi inizialmente possibili; 7) Il teorema di Bayes è d'altronde indispensabile in caso di contenzioso al fine di misurare la responsabilità dell'errore medico quale fattore causale del danno, tenendo conto del peso dell'evidenza a disposizione.*

Parole Indice Metodologia clinica, Teorema di Bayes

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Introduction

Handbooks of inductive logic, often use medical examples in order to exemplify how the uncertainty affecting medical diagnosis can be quantified and managed through computation methods developed out of probability theory (Bermudez 2009; Hacking 2001; Mushlin and Greene 2010; Peterson 2009). In these examples, epidemiological data about the incidence of a given disease on a population of interest, are combined with symptomatic evidence and laboratory data in order to provide an individual diagnosis or risk assessment. The result is a ranking of possible diagnoses according to the probability associated with each of them. In the ideal case, one diagnosis is given probability 1 and the others 0. In the case of maximal uncertainty, each hypothesis is given the same probability ($P = 1/n$, where n is the number of the hypotheses under consideration). Probabilistic expert systems represent the computational application of this approach to diagnosis (see for instance Cowell et al., 2007).

On the other hand, traditional handbooks of clinical diagnosis, especially differential diagnosis, rather focus on the semeiotic work that doctors are supposed to perform in order to progressively reduce the spectrum of candidate hypotheses and finally, by a work of systematic elimination, arrive at the hypothesis, which the available evidence fails to eliminate (Blois, 1984; Burnum, 1993; Kassirer, 1989; Kassirer and Kopelman, 1991). This work resembles that of the (hypothetical-) deductive method, where evidence can only exclude the contemplated hypothesis but has neither the capacity to confirm it nor to strengthen or weaken it (Popper, 2002). Being inherently deductive, this methodology seems to be hardly reconcilable with the probabilistic method mentioned above.

Indeed, apart from the inference direction – top-down in the deductive paradigm vs. bottom-up in the inductive one – there is another fundamental difference between the two models: in the Bayesian one, there is a closed set of mutual exclusive hypotheses, whose probability-sum equals 1 (the so called "sample space"). This model determines straightforward knowledge updating on the basis of available evidence, where probability increase of one hypothesis necessarily leads to probability decrease of at least one of the others. It's a sort of cake-diagram epistemology, where the main constraint for rationality is that, increase in the confidence about one hypothesis, directly diminishes confidence in the others.

In the hypothetical-deductive method instead, there is no formal constraint relating the plausibility of one hypothesis to the plausibility of another: exclusion of one hypothesis (or a set of hypotheses) does not necessarily lead to any change in the confidence with which any other alternative one is entertained. What happens instead, is that a new hypothesis must be conjectured on the basis of the knowledge provided by the failure of the previous one to pass the test.

Yet, another form of inference which is relevant in the domain of medical reasoning, as well as in that of scientific explanation, is that of "abduction". This term has been introduced by Charles Sanders Peirce in "Deduction, Induction and Hypothesis" (1934, Collected Papers 2.623) and further developed in the Cambridge Conferences (1898) and in the 1903 Harvard Lectures. Following Peirce, abduction can be considered to cover two types of inference: the classification of a token under a certain predefined class (also known as qualitative or analogical induction), or the invention of a hypothesis for explaining a "surprising" fact. In this second sense, abductive inference covers the etimological sense of intelligence as the act of connecting disparate things (inter-ligere = "bind between") and is grounded on causal thinking. Instead, the former form of abduction rests on a semantic level, in the sense that it acts as a function which maps individuals to their class.

In the following, all these different forms of inference are examined as diagnostic tools in medicine. A case history will show that each of them bring a distinct contribution to the final diagnosis, thereby demonstrating that they are, at least practically, reconcilable.

Deduction

In deductive inference, the conclusion follows necessarily from the premises, in that it makes explicit the information already contained in them. It should be noted that even if totally based on already available information, deduction may deliver new knowledge. As a matter of fact, deductive inference makes explicit what is implied by the *conjunction* of the premises; moreover, this process produces awareness about their intimate connection and related implications thereby revealing information about relationships.

In medical diagnosis, deduction can be used whenever a necessary and/or sufficient connection between evi-

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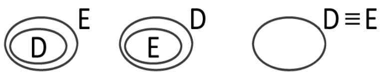
dence (E) and disease (D) has been established. This connection can be represented by the entailment relationship as follows:



	D	E	D→ E	E → D	D↔ E
1	T	T	T	T	T
2	T	F	F	T	F
3	F	T	T	F	F
4	F	F	T	T	T

Table 1: Venn diagrams and corresponding truth tables for different entailment relations between D (diseases) and E (evidence).

The case where the contemplated disease D implies the observed evidence E ($D \rightarrow E$), can represent the relationship of *causal sufficiency* between D and E, meaning that D is a sufficient (but not necessary) cause for E;

Instead the opposite entailment relationship, $E \rightarrow D$, can represent D as being a *necessary cause* for E (wherever E is present, then D must be too);

In the same line, $D \leftrightarrow E$ can be interpreted as meaning that D is a necessary and sufficient cause for E.

By knowing that $D \rightarrow E$, and that D is given, one can *predict* that the observable phenomena E will follow:

$D \rightarrow E$
D
E

By knowing that $E \rightarrow D$, after observing E, one can *diagnose* that H is the case:

$E \rightarrow D$
E
D

By knowing $D \leftrightarrow E$, one can both infer D from E and E from D.

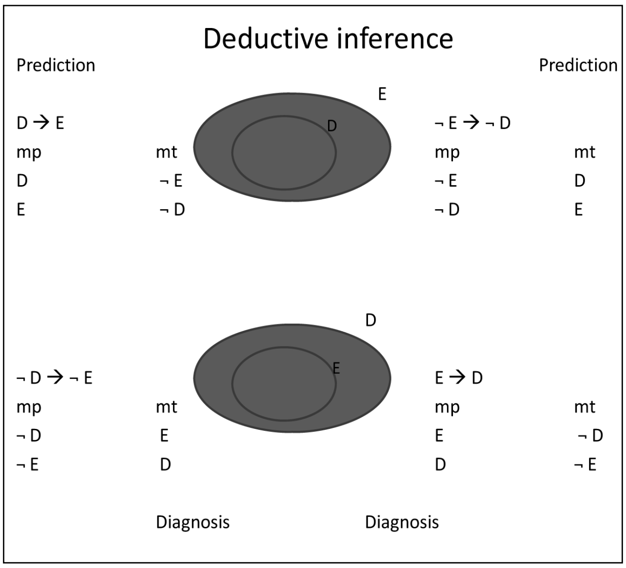
Provided that diagnosis consists in the interpretation of symptoms and other laboratory or biomedical data, the route goes necessarily from evidence E to diagnosis D and therefore, the only deductive inferences which can be made, are *modus ponens* when the relationship between E and D is $E \rightarrow D$ (e.g. whenever the symptom E is present, then, disease D is necessarily present):

$E \rightarrow D$
E
D.

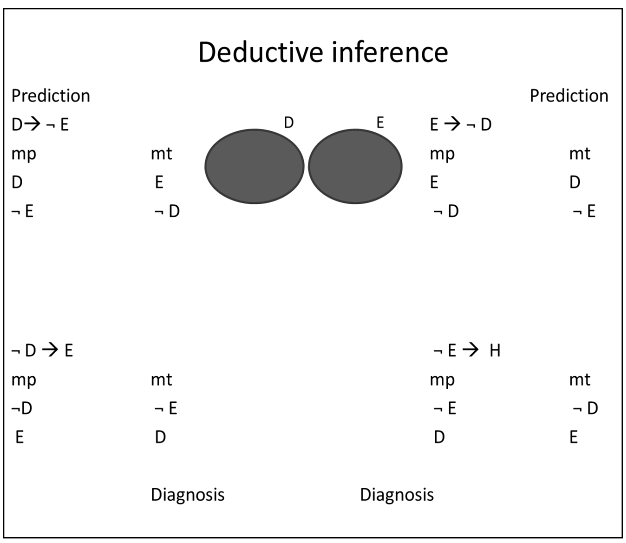
or *modus tollens* from negative evidence and knowledge of a relationship of causal sufficiency between D and E:

$D \rightarrow E$
$\neg E$
$\neg D$.

In this second mode of reasoning, evidence can only contribute to exclude one hypothesis. The table bellow represents the possible strategies of deductive inference, given a positive causal relationship between D and E (mp stands for *modus ponens* and mt for *modus tollens*):



Deductive inference can also follow from the premise $D \rightarrow \neg E$ (or equivalently $E \rightarrow \neg D$), and $\neg D \rightarrow E$ (or $\neg E \rightarrow D$); e.g. when the causal relationship is negative or D and E exclude each other. In this case, the possible inferences are illustrated below:



Strict deductive inference however is rarely justified by medical knowledge, which is notoriously affected by endemic uncertainty due to the ambiguity of symptoms: different diseases show similar symptomatic configurations (the same symptom can be present in different diseases) and the same disease may show different configurations of symptoms from case to case. Moreover, disease classification is very complex: some diseases are very well known in both etiology and form, others are best addressed as syndromes; e.g. sets of concomitant symptoms which happen to be recurrently observed together, but for which there is little comprehension of the underlying phenomena. In these cases, the symptoms themselves are the illness. There is a continuum of examples between these two extremes.

Because of the complexity that characterizes the relationship between observable evidence and disease, medical reasoning tends to be modelled through inductive rather than deductive paradigms. Moreover, whereas inductive reasoning can be both qualitative and statistical, it generally tends to be equated with the latter form (probabilistic induction) thereby neglecting the possibility of analogical induction or other forms of inference (such as argumentative: see Fox, 2000, 2003, forthcoming). This raises two distinct questions, which will not be dealt with here: the first one concerns the descriptive adequacy of the inductive-probabilistic model against other ones; the second issue concerns its habitual priority in respect to other methods of diagnosis.

The aim and purpose of this paper (which is descriptive rather than evaluative) do not allow me to consider this question in detail; instead I will go on to present the customary standard view of inductive reasoning.

Induction

Whereas deductive inference follows necessarily from the information assumed in the premises, induction aims to gain new knowledge from observation and/or experimentation or through analogy.

Induction departs from deduction mainly for two kinds of steps, which are fallacious from a strictly deductive point of view.

1. The inference *tries to establish the connection between D and E* instead of drawing a conclusion from it. Logically speaking, there is no way to arrive at $D \rightarrow E$ (or vice versa) by simply observing the co-occurrence of D and E events (Hume docet)[1]. For this reason the relationship itself is hypothetical and henceforth the alternative diagnostic hypotheses will be denoted by the letter H (for hypothesis).
2. Once you have $D \rightarrow E$ or you have inductively established a relationship between H and E, one infers, by observing E, that D (or H respectively). This step is a fallacy from a deductive point of view (fallacy of affirming the consequent) and is also called inverse induction (more on this in the next paragraph).

Point one consists, for instance, in drawing the conclusion that "All swans are white" ($S \rightarrow W$) after observing a sufficiently large and representative sample of n white swans. This is called general enumerative induction. Particular enumerative induction refers to the prediction that a particular individual will follow a certain law, based on observation of n individuals of the same class: "the next swan will be white".

This mode of reasoning has two corollaries: 1) the link direction cannot be established by the association itself; 2) the strength of the association says nothing about the kind of connection (e.g. causal). The only inference which statistical observation can legitimize, is that of dependence/independence of types of events (this point is however an issue of heated debate in the philosophy of science. See for instance Cartwright 2007 and Glymour, 2009).

Inductive inference can work in different directions (for instance, from sample to population, from sample to individual or from population to sample) depending on which data are available and which questions one wants to answer. Furthermore, it can follow from an analogical procedure (qualitative induction)[2] or be based on counting (enumerative induction; see Kyburg, Man Teng, 2001 for an introduction).

Inverse induction

Through direct induction, a property about a certain class of individuals is inferred from observation of a representative sample (see the above swans' example). Instead, inverse induction is a (possibly probabilistic) inference concerning a single case, based on statistical or other kind of information.

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[1] The issue is however not settled yet. Already soon after Hume's refusal of causal laws, Bayes and Laplace have formulated the so called Rule of Succession, precisely to defend the capacity of induction to bring new valid knowledge through accumulation of evidence (see also Huemer, 2009).

[2] Qualitative (or analogical) induction follows from comparing individuals or classes and then inferring a property of one from the collection of properties associated with another one. For instance: if disease A is generally associated with symptoms α, β, γ, δ and disease B seems to show a similar etiology, then B is also supposed to produce symptoms $\alpha \vee \beta \vee \gamma \vee \delta$. Conversely, by observing an individual with symptoms α, β, γ and δ, one will infer that the individual may have disease A. This second type of inference is called inverse induction.

Inverse *qualitative* induction goes from the properties of the individual to its class.

Observation:	a has property α, β, γ and δ (individual a has property α, β, γ and δ)
Rule:	x belongs to A → x has property α, β, γ and δ (if an individual x belongs to class A then it has property α, β, γ and δ)
Conclusion:	a belongs to A. (therefore, individual a belongs to class A).

In this kind of inference knowledge about classes (e.g. the properties of their elements) is used to identify the class a specific observed individual belongs to. In this case, for instance, observation of the properties of an individual (α, β, γ and δ) triggers the search for the class whose individuals share the same properties: A = {x|x has property α, β, γ and δ}. From this follows the entailment rule: x belongs to A → x has property α, β, γ and δ. But the conclusion is fallacious. In fact, the inferential step would be legitimate only when the entailment would go in the opposite direction: x has property α, β, γ and δ → x belongs to A.

This becomes clearer when comparing inverse induction and deduction:

Deduction

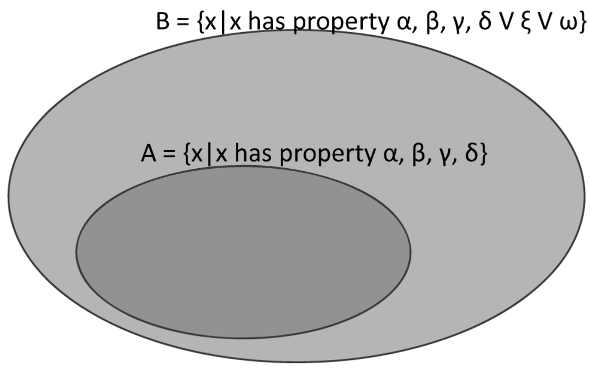
Rule:	If x belongs to A → x has properties α, β, γ and δ
knowledge:	x belongs to A
Conclusion:	x has properties α, β, γ and δ

Inverse induction

Rule:	If x belongs to A → x has properties α, β, γ and δ
Observation:	x has properties α, β, γ and δ
Conclusion:	x belongs to A

Logically speaking, having properties α, β, γ and δ is a necessary consequence of belonging to class A, but the reverse does not hold: belonging to A is not a necessary consequence of having properties α, β, γ and δ. Therefore, one cannot infer the class to which an individual belongs to from knowledge about its properties, unless the set of properties defines that class uniquely (which case would be represented by a biconditional relationship ↔ not by a simple sign of entailment).

Let's show this graphically through a Venn diagram: set A is the set of all individuals sharing properties α, β, γ and δ; set B is the set of both individuals sharing properties α, β, γ, δ and individuals having properties α, β, γ, δ plus additional properties (say ω and ξ). Then properties α, β, γ, δ would both indicate disease A and disease B:



Medical diagnosis is generally based on the fallacy of moving from the consequent (the observable symptoms and/or lab data) to the antecedent (the diagnosis). However, given the constraints which characterise the medical setting, and the regular unavailability of perfect diagnostic information, one is obliged to make virtue of necessity and use this kind of inference in such a manner that one comes as close as possible to the right diagnosis and therapy.

It is thus "logical" that Bayes' theorem, which mathematically formalizes this type of inference and quantifies the uncertainty surrounding it, has been called to rescue as an aid to reduce diagnostic errors by changing the inference from a qualitative into a quantitative one and by providing an explicit assessment of the probability which can be assigned to each hypothesis on the basis of available evidence. In fact, if it is true that both quantitative and qualitative induction are affected by uncertainty, nevertheless in the case of quantitative inferences, the uncertainty itself can be quantified and better tracked along the paths.

Bayes ' Theorem

The main use of Bayes' theorem in medical diagnosis is to assess the probability that an individual belongs to a certain pathological profile, given the available evidence.

The special merit of Bayes' theorem lies in the fact that ambiguous evidence - such as symptoms which are expressed by more than one disease - is modelled in a likelihood function which provides information about how strong the evidence is associated with one candidate rather than the other.

The likelihood function expresses the proportion of joint (H_i & E) cases over the entire quantity of Es for each alternative hypothesis $H_i = (H_1; H_2; \dots H_n)$. This function may be based on the information provided by epidemiological, clinical and other relevant data. Bayes' theorem then applies it within the framework of the probabilistic calculus in order to update knowledge about the set of hypotheses.

An example might illustrate how. Imagine that a symptom S is strongly connected to a specific illness A (and very weakly to other diseases), then the presence of this symptom in a diagnostic procedure, strongly favours the medical diagnosis towards this illness. Bayes' theorem may help quantify how much knowledge updating is justified in the light of the presence of the symptom.

In probability terms, we could assume that the likelihood of a certain disease A on a symptom S, is very high, say in .95. This means that 95% of the cases where the disease is present also S has been observed. Similarly for the likelihood of other diseases (B, C, D):

P (S/A) = .95
P (S/B) = .05
P (S/C) = .02
P (S/D) = .03

If the doctor is expert enough to judge that no other hypotheses can be taken into consideration, and that the possibility of the conjunct presence of more than one illness at a time is negligible (which equals to say that they are altogether exhaustive and mutually exclusive), then Bayes' theorem applies. In a situation where the doctor is uncertain about illnesses A through D, the occurrence of such a symptom would enormously increase the probability of A against the others even if it is epidemiologically very rare. If epidemiological data about the incidence of the diseases are available, for instance: P(A) =.01, P(B) =.35, P(C) =.5, P(D) =.14, than the probability of the conjunction of symptom and illness would be respectively:

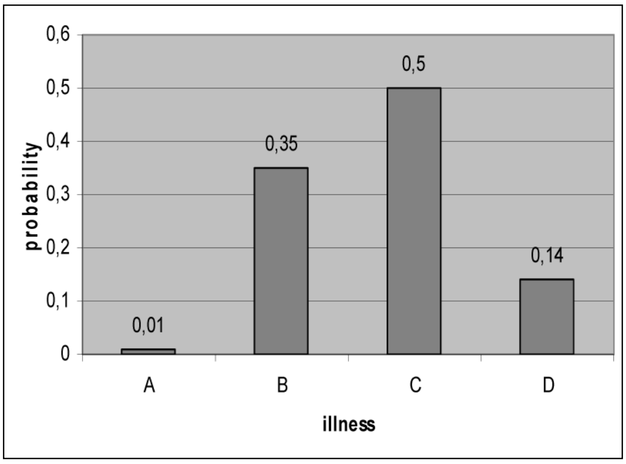
P(A & S) = P(A) x P(S/A) = .01 x .99 = .099
P(B & S) = P(B) x P(S/B) = .35 x .05 = .007
P(C & S) = P(C) x P(S/C) = .5 x .02 = .025
P(D & S) = P(D) x P(S/D) = .14 x .03 = .0042

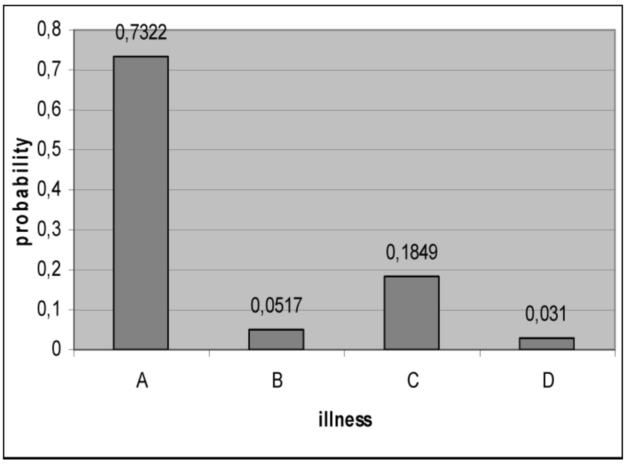
Which gives the absolute probability of S: P(S) = P(A & S) + P(B & S) + P(C & S) + P(D & S) = .1352

The probability of each illness *given the information provided by the symptom* is computed out of the ratio of each conjunct over the probability of the symptom = P (I & S)/P(S), (where I stands for any of the illness considered):

P(A/S)= .099/.1352 = .7322
P(B/S) =.007/.1352 = .0517
P(C/S) = .025/.1352 = .1849
P(D/S) = .0042/.1352 = . 0310

Therefore the symptom information has radically changed the probability distribution of illnesses A through D from: .01; .35; .5; .14 to .7322; .0517; .1849; .0310 respectively. This can be shown graphically:





As the graphs show, the prior distribution tends to be rather diffuse, whereas the posterior distribution has a visible peak on value A, which represents a certain degree of uncertainty reduction in relation to the possible state of affairs. In this case, moreover, the mode of the probability distribution radically shifts towards the value, which in the prior had the minimal frequency. This effect shows the great impact of the information provided by the symptom. Not every piece of information has a comparable effect though, and sometimes sample information can even increase uncertainty if it "levels out" all frequencies instead of favouring some of the parameter values against the others. For example, an ambiguous symptom such as cough (which is loosely connected to several afflictions) might be of little help for a diagnostic assessment.

Bayes' theorem says nothing about the type of semiosis between evidence and hypothesis (whether causal-indexical, symbolic or iconic, to use Peirce's semiotic taxonomy); however, by providing a measure of the likelihood of each entertained hypothesis on the available evidence, it allows to draw inferences which on a pure-ly categorical basis could not be warranted. This is because, by working in a probabilistic framework, it makes explicit the level of confidence with which a hypothesis is entertained without being forced to commit itself to its truthfulness or falseness.

Another considerable advantage of the Bayesian paradigm is that it allows translating the epistemic value of evidential information directly into practical advices. Furthermore, the practical value of incoming information can be evaluated through so called "sensitivity analysis". This analysis allows to predict the bearing of further information on the decision at hand as a function of its capacity to change the options ranking. This is a relational capacity, which depends not only on the information's relevance, but also on the intensity of preference between outcomes.

The major epistemic advantage of the Bayesian paradigm however, is that it models uncertainty as a function of "equivocation" between hypotheses: the closer the probabilities assigned to each hypothesis, are to each other, the more uncertain is the diagnosis. The epistemic value of evidence ("relevance") is greater where it points to one hypothesis rather than others and, thereby, both increases its probability and decreases the probability assigned to the others.

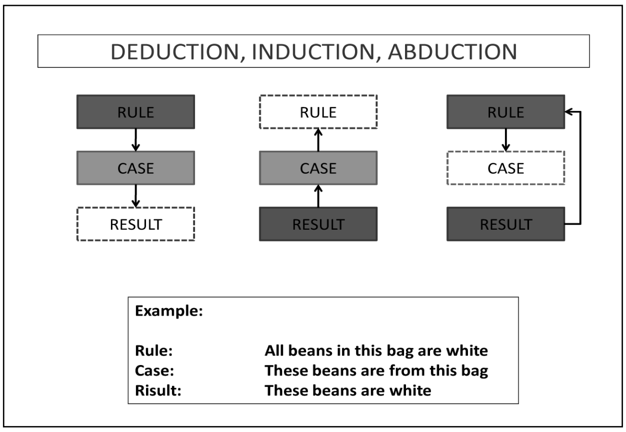
Additionally, uncertainty may be modelled in the Bayesian paradigm also by allowing a partition cell devoted to the vague hypothesis: "other". The greater is the value of this cell, the greater is the portion of ignorance affecting the diagnosis and the related decision.

In summing-up, the Bayesian paradigm not only makes the portion of "not-knowledge" explicit, but it provides a framework where ignorance is, in some sense, quantified (and therefore tracked or "controlled for").

Abduction

Charles Sanders Pearce has introduced the term abduction in different contexts, with different semantic nuances (see Thagard 1988, § 4.2.1).

A first meaning of abduction can be derived from the following comparative table.



The table is a revised version of the synopsis offered in "Deduction, Induction and Hypothesis" (Peirce, 1934, CP: 2.623; see also Petoefi, 2000 and Vitalcolonna, 1999). It illustrates the different step order performed by deductive, inductive and abductive (or "retroductive") inference. The well-known example proposed by Peirce is:

(1) Deduction:
All beans in this bag are white;
These beans are from this bag;
These beans are white.

(2) Induction:
These beans are white;
These beans are from this bag;
All beans in this bag are white.

(3)Abduction (or inverse induction, or "retroduction"):
These beans are white
All beans in this bag are white
These beans are from this bag.

In the case of deduction, from a rule: "If x belongs to A then x has property T", and a case: "a belongs to A", a result is inferred which is necessarily true given the premises: "a has property T".

In the case of direct induction, empirical observations about a phenomenon are used to support a generalisation that translates into a rule: the observation of a certain sample (all the beans drawn from the bag are white) constitutes the basis for an ampliative inference.

In the case of abduction, the observation of a phenomenon *and* knowledge of a rule allow for the classification of the phenomenon under the case predicted by the rule.

Whereas the deduction correctly uses *modus ponens*, both induction and abduction are fallacious inferences. However, they are intuitively plausible. Induction generalises a property from a sample of the population; whereas abduction uses a general rule to explain an observed fact by subsuming it under a case of the rule. In this respect, abduction corresponds to the kind of inverse induction (either quantitative or qualitative) introduced in the preceding paragraph.

The parallelism between deduction and abduction is due to the fact that both use type-token information. However, the inference direction is opposite: whereas deduction uses information about the type in order to assert something about the token; abduction uses information about the token in order to identify the type to which it belongs. Direct induction instead, is mainly occupied with the establishment of this relationship itself.

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The Bayes' theorem presented above does nothing else than quantifying the uncertainty surrounding the abductive leap: a phenomenon E is associated with rule H (with probability p) and whenever E is observed in a specific case, it contributes to classify this case as a token of H with a degree of probability which follows from the likelihood of H on data E.

The rule could be interpreted, in Bayesian terms, as the degree of association between evidence and entertained hypothesis: P (E/H); the observation is simply E; finally, the conclusion is the posterior probability that H is indeed the case, given that E has been observed: P(H/E):

Rule → P(E/H)
Evidence → E
Conclusion → P(H/E)

Inference steps	Deduction	Inverse induction (qualitative)	Inverse induction (quantitative – probabilistic)
Rule	All beans in this bag are white	All beans in this bag are white	100% of beans in this bag are white.
Evidence	These beans are from this bag	These beans are white	There are n white beans on the table.
Conclusion	These beans are white	These beans are from this bag	There is a probability P that the white beans come from this bag (rather than from somewhere else).

It is important to note that whereas, deduction only focuses on one hypothesis and considers whether it is true/false and qualitative inverse induction tries to work out the most plausible hypothesis given the observed facts, Bayes' theorem requires the likelihood of *all alternative hypotheses* on the available evidence to be made explicit, in order to compute the posterior probability of any hypothesis given the observed evidence. In the beans' example for instance, in order to compute the probability that the beans come from the bag, one needs to know the likelihood that a sample of n white beans are found on the table, given that they *do not come from that bag*. Similarly, in the symptom-diseases example, provided above, S is the symptom which is differently associated with each of the contemplated diseases (the hypotheses under consideration); by using knowledge that S is the case, the doctor can update his diagnosis about the patient's disease with a degree of probability which depends on the strength of association between S and the different diseases under consideration:

Inference steps	Inverse induction (quantitative – probabilistic)
Rule: $P(E/ H_i)$	P (S/A) = .95 P (S/B) = .05 P (S/C) = .02 P (S/D) = .03
Evidence: E	S
Conclusion: $P(H_i /E)$	P(A/S) = .7322 P(B/S) = .0517 P(C/S) = .1849 P(D/S) = . 0310

Abduction II: Hypothesis invention

The term abduction, is used in Peirce's work also in another sense. Beyond denoting the act of classifying a token under a predefined type – as presented in the preceding section, abduction also refers to the conception of a new hypothesis in order to *explain* the observed ("surprising") state of affairs: abduction "is where we find some very curious circumstance, which would be explained by the supposition that it was a case of a general rule and, thereupon, adopt that supposition" (Peirce, 1878: 2.624). Using the beans' example, the inferential steps could be written as follows:

Evidence:	The beans are white;
Knowledge:	There is a bag in this room whose beans are all white;
E & K → H	
(explanation):	The fact that the observed beans are white, and that there is a bag containing only white beans, can be explained by the hypothesis that the beans come from this bag;
Conclusion → H:	The beans come from this bag.

In this sense, abduction is a form of inference which pertains rather to hypothesis invention, than to hypothesis confirmation (or refutation). One observes E and knows K and tries to find out a hypothesis which can explain the occurrence of E in the light of K. Peirce formulates this form of abduction as follows:

The surprising fact C is observed;
But, if A were true, C would be a matter of course,
Hence, there is a reason to suspect that A is true.
(Peirce, 1878: 5.171).

Abduction of the first kind works on a semantic level: e.g. it aims to give a name to a phenomenon which has already been categorized under a specific heading. Instead, abduction of the second type uses causal reasoning in order to explain facts in the light of available knowledge.[3]

Moreover, the starting point and conclusion of the two types of abduction are opposite:

1. Whereas the focus of abduction I is on the capacity of the observed evidence to strengthen or weaken a hypothesis; abduction of the second type starts from the data and looks for a hypothesis which makes their *conjunction* plausible;
2. Whereas in abduction I the inference *is based on* a (probabilistically) established association; in abduction II *the association itself is the goal* of the inference.

In medical diagnosis abduction II might come to its own when evidence is so contradictory that the case cannot be categorised under any of the already known disease classes and a new typology or sub-typology needs to be invented in order to account for the set of symptoms presented by the case at hand.

Hypothetical-deductive method

The hypothetical-deductive method combines deductive inference with the second type of abduction. In practice, it tests the hypothesis inferred through abduction on the basis of its observable implications. It fundamentally applies *modus tollens*:

1.	E	evidence E is observed;
2.	K	relevant knowledge K is considered;
3.	E&K → H	the conjunction of E and K indicate that H should be the case;
4.	H → I	H entails implication I;
5.	¬I	experiment shows that I is not the case;
6.	¬H.	therefore, also H is not the case.
7.	G	a new hypothesis G must be conceived in order to account for E, K, and ¬I.

A set of data (evidence E), together with a certain amount of knowledge about a phenomenon, indicates that H should be the case. In order to see whether this is really so, one looks for a necessary implication of H and put up an experiment in order to test whether it holds. By doing this, one tests the hypothesis H. However, the test can give only negative conclusive information, e.g. it can say that ¬H, given that ¬I is found. Every time that ¬ I is found, the hypothesis H needs to be refined or completely changed. Instead, if I is found to be true, then H cannot be said to be confirmed but only "corroborated". This means that in a set of hypotheses, those that have been submitted to more tests, and passed them, are considered more plausible than those that have not been yet refuted as well but have passed less and/or less severe tests (Popper, 2002).

In medical diagnosis, the hypothetical-deductive

[3] Abduction II can be considered to be equivalent to what epistemologists have been studying also under the name of "inference to the best explanation". There has been however a coalescing of both types of abduction concepts into one, in the debate around the comparison between Bayesian inference (i.e. abduction I probabilized) and inference to the best explanation (i.e. abduction II) (see among others McGrew, 2003; Huemer, 2009; Lipton 2004, Psillos 2004, Salmon 2001a, 2001b, Weisberg 2009) which has compromised the clarity and fruitfulness of the dispute in many ways. An interesting dyad emerging from the discussion is the opposition between evidential and theoretical consilience (see McGrew 2003) which provides an attractive – although not unproblematic – interpretation of epistemological coherence (more on this in a forthcoming paper; see also Bovens and Hartman, 2003).

method is represented by cases where the doctor seeks for plausible diagnostic hypothesis and test them by looking at evidence which can exclude them one after the other. The sophistication of this process depends on the doctor's knowledge of the possible implications of any disease either in terms of observable symptoms or in terms of laboratory data.

The formal difference between a straightforward *modus tollens* and a hypothetical deductive one in medical diagnosis, is that, in the former, both hypotheses and possible implications are known, whereas in the latter, at least one of the two must be thought of anew during the diagnostic process.

The difference between abduction II and the hypothetical deductive method, is that, while abduction II stops at stage 3 of the inferential process illustrated above, the hypothetical-deductive method tries to falsify the inferred hypothesis through *modus tollens* (steps 4-6).

Until now, I have presented a series of inferential methods, which have been included to a lower or higher degree in the toolkit of medical reasoning. Indeed, medical diagnosis seems to combine different kinds of inference in subsequent steps depending on the available evidence. In the following, I present a case history in order to show what types of inference models are used in different diagnostic circumstances and the rationale underlying the inferential strategy.

A case history: Frugoni's diagnosis of miliary tuberculosis

I am reporting here an old case history from the famous Italian clinician Cesare Frugoni, who wrote one of the most used manuals of differential diagnosis in the first half of the XXth century: "*Lezioni di clinica medica*" (1948). Federspil and Vettor have already examined the case, and, although I fundamentally agree with their analysis, I will propose some additional comments and come to a different conclusion.

The clinical case reports a diagnosis of acute miliary tuberculosis (or disseminated tuberculosis). Miliary tuberculosis is the most dangerous type of tuberculosis, because the infection is not limited to one organ (e.g. lungs or kidney), but it spreads throughout the body through blood circulation.

Given that generally infections have a short incubation time, patients affected by infectious pathogens only, slowly show noticeable signs of the disease. In fact, the central figure of the case, a young woman, presents practically no laboratory or symptomatic evidence. She has been taken to the hospital because of two episodes of severe fever interspaced by a two-week-period of relative well-being.

The first fever was accompanied by skin eruptions on the legs: big whitish-bluish infiltrated nodules. Frugoni interpreted these as "clear signs" of Erythema nodosum. Erythema nodosum however, is not a disease, but a syndrome with various etiologies: in fact, it can be caused either by toxic agents or rheumatic infections, or else, by the use of sulfonamides. In most cases however, it is an allergic reaction to tubercular infections. Frugoni quotes a personal statistics from his clinic, where 94% of EN has been established to be of tubercular origin. The link between EN and tuberculosis is the key to the general diagnosis. However, let's follow Frugoni's line of reasoning. The table presents, in the first column the observed data; in the second one, the diagnostic question and the provisory hypothesis; in the third column, the inference steps that lead to the conclusion.

1

Observation (E)	Hypothesis (H)	Inference steps	
Fever	Cause?	Rule:	Fever → Infection (if fever then infection)
		Observation:	Fever
		Conclusion: (modus ponens)	Infection

The first symptom analysed is the presence of fever; this straightforwardly indicates the presence of an infection, given that there is a necessary causal association between fever and infection. The second step then, is to diagnose the type of infection.

The patient's general state, at the moment of admission in the clinic, is relatively good, apart from her high temperature and symptoms of asthenia, weakness and swollen spleen and liver. Both physiological and instrumental evidence is negative (which is obviously different from saying that there is no evidence): no lesions at the lymphoglandular system, nothing at the head, no headache, no signs of psychic disorder, no neck-stiffness, nothing to the chest (heart, lungs). As for the instrumental evidence, radioscopy is negative, but the pulsation is abnormal and there is light leucopenia. The available evidence excludes that the fever can be originated by lesions at the gallbladder by a pleurisy, by pneumonia or by pararenal abscess. All these negative signs indicate that the infection is not specific, and therefore, that it is generalised to the entire body:

2

Observation (E)	Hypothesis (H)	Inference steps
¬ abnormal signs at lymphoglandular system; head; neck; chest; lungs; heart; negative radioscopy	What kind of infection?	Rule: Specific infection → (lymphoglandular system V head V neck V chest V lungs V heart) Observation: ¬ LS ¬ head ¬ neck ¬ chest ¬ lungs ¬ heart Conclusion: ¬ specific infection (modus tollens) Rule: For all x (x is an infection) → x is specific V x is generalized Observation: q is ¬ specific Conclusion: q is generalized

The swollen liver and spleen, usual expression of a general infective state, also indicate the non-specificity of the infection. This is however inconclusive evidence, because swollen liver and spleen can also be caused by other phenomena. Therefore, the causality connection between general infection and swollen liver and spleen, is sufficient but not necessary: GF → SLS but not SLS → GF. Hence, concluding GF from GF → SLS and the presence of SLS is a fallacy. But, from an inductive point of view, it can be considered as a case of abduction of the first type.

3

Observation (E)	Hypothesis (H)	Inference steps
Swollen liver and spleen	What kind of infection?	Rule: General infection → Swollen liver and spleen Observation: Swollen liver and spleen Conclusion: General infection (Abduction I)

Now, the question is: What kind of general infection? Candidate diseases for the explanation of a general infection with swollen liver and spleen, are the Malta-fever, slow endocarditis, malaria, typhus, sepsis and tuberculosis: the set of hypotheses is therefore represented by the disjunction: {Malta-fever V slow endocarditis V malaria V typhus V sepsis V tuberculosis}.

Typhus is quickly excluded because Typhus is associated to Eberthian-fever, rosy elements, chest erythema, neurological syndrome, intestinal syndrome and specific immune reactions. Neither of these phenomena appear to affect the subject under examination.

4

Observation (E)	Hypothesis (H)	Inference steps
¬ Eberthian-fever, ¬ rosy elements, ¬ chest erythema, ¬ neurological syndrome, ¬ intestinal syndrome ¬ specific immune reactions	What kind of general infection?	Rule: Typhus → (Eberthian-fever V rosy elements V chest erithema V neurological syndrome V intestinal syndrome V specific immune reactions) Observation: ¬ ef ¬ re ¬ ce ¬ ns ¬ is ¬ sir Conclusion: ¬ Typhus (modus tollens)

Sepsis is also excluded, because when someone has swollen liver and spleen because of sepsis, then he also has septic tongue, neutrophilia and is in a numbed state. Instead, the patient is lucid and calm, has leucopenia, and no septic tongue. Therefore, the infection is not a sepsis:

5

Observation (E)	Hypothesis (H)	Inference steps
a has no septic tongue a has leucopenia a is lucid and calm	What kind of general infection?	Rule: Sepsis → septic tongue V neutrophilia V numbed state Observation: ¬ septic tongue Leucopenia → ¬ neutrophilia Lucid and calm → ¬ numbed state Conclusion: ¬ sepsis (modus tollens)

Malaria is excluded because the malaria's parasite test is negative, and endocarditis is also immediately excluded given that there are no heart abnormalities:

6

Observation (E)	Hypothesis (H)	Inference steps
Negative test result for malaria parasite	What kind of general infection?	Rule: Malaria → parasite Observation: ¬ parasite Conclusion: ¬ malaria (modus tollens)
Heart = normal	What kind of general infection?	Rule: Endocarditis → heart abnormalities Observation: Heart = normal Conclusion: ¬ endocarditis (modus tollens)

As for the Malta-fever, instead, there are *signs in favour of a diagnosis* of this type: these, are the swollen spleen and liver, the irregular and instable temperature, the presence of leucopenia and the abundant sweating. However, there is also evidence against the hypothesis of Malta-fever: both blood culture and serum reactions are negative. Frugoni concludes that '*It is therefore probable that it is not Malta-fever*'. It is important to note that the probabilistic conclusion is due to two different constraints:

1. On the one hand, evidence in favour of the hypothesis cannot be conclusive because it is positive and, therefore, *modus tollens* cannot be applied. The relationship between the observed symptoms and the diagnostic hypothesis is not necessary: Malta-fever is a sufficient cause for irregular and instable temperature and so on, but it is not a necessary one, so that the evidence cannot be used to infer the diagnosis deductively.
2. On the other hand, there is also inconclusive evidence *against* the hypothesis of Malta-fever: the negative blood spectrum and the negative serum reaction. These two test results cannot absolutely exclude the presence of the disease, and therefore, also here, *modus tollens* cannot apply, but the data obviously speaks against it.

7

Observation (E)	Hypothesis (H)	Inference steps
Inconclusive evidence for MF Irregular and instable temperature Swollen spleen and liver Leucopenia Abundant sweating *Inconclusive Evidence against* MF Negative blood culture Negative serum reaction	What kind of general infection?	Rule: Malta-fever → irregular and instable temperature V swollen spleen and liver V leucopenia V abundant sweating Observation: Irregular and instable temperature Swollen spleen and liver Leucopenia Abundant sweating Conclusion (only on probabilistic grounds): low probability of Malta-fever.

The final diagnosis of miliary tuberculosis comes by exclusion of the alternative hypotheses and it is additionally supported by further radiological evidence: lung parenchyma (the radiography shows the lungs full of little "seeds" which are typical of tuberculotic caseation). As a further confirmation, the same "seeds" can also be observed in the patient's cornea. The following table illustrates the inferential process that results in the final diagnosis.

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8

Observation (E)	Hypothesis (H)	Inference steps
Erythema nodosum Seeds in the cornea Seeds in the pulmonary radiography	What kind of general infection?	Rule: general infection & swollen liver and spleen → Malta-fever V endocarditis V malaria V typhus V sepsis V tuberculosis. ¬ endocarditis ¬ malaria ¬ typhus ¬ sepsis ¬ MF (?) + statistical evidence concerning the etiology of erythema nodosum + radiography + symptomatic evidence in the cornea Conclusion: miliary tuberculosis

The inference process goes from a very general assessment ("infection") and progressively specifies the pathological classification. Inference steps are generally made through *modus tollens* until the Malta-fever hypothesis is both supported and disconfirmed by inconclusive evidence and therefore, cannot be categorically excluded. The final preference for the tuberculosis hypothesis against the Malta-fever, is the result of a comparison of the evidence for and against both of them.

In this diagnosis, Frugoni represents medical reasoning as mainly deductive. However, this is also due to the didactic exposition of the case and the need of justification of the final diagnosis. Between the lines, one can read an overarching inferential structure, which originates from the *puzzling conjunction of the fever, and the absence of any symptomatic evidence or laboratory report* ("there is no semeiotic fact which can explain by itself the presence of fever"): all localised lesions (like appendicitis, for instance) which can give origin to fever, are absent.

Given that there is no other possible explanation, the fever is attributed to a general state of infection (abduction II), but this diagnosis is very vague and does not give any information about the etiology. This is investigated by taking into account all possible types of general infections that are accompanied by swollen liver and spleen. In order to simplify the diagnostic procedure, conclusive negative evidence for each of them is examined in order to exclude each of them, one after the other. This procedure simplifies the inferential process by considering one hypothesis at a time; however, it is possible only when the relationship between evidence and disease is necessary in one direction or the other.

A probabilistic paradigm is used only when no conclusive negative evidence can be used to reject the hypotheses under consideration (here the Malta-fever), in which case, the non-rejected hypotheses are weighed against each other on the basis of all available evidence. In this case, tuberculosis wins over the Malta-fever.

Frugoni takes into account also the epidemiology of erythema nodosum (94% of cases in his clinic has been established to be caused by tuberculosis). It is important to note, that this data has been reported both at the beginning and at the end of the diagnosis. In fact, the presence of this syndrome seems to have covertly guided the entire inferential procedure towards the final diagnosis. Because of its diverse etiology, it could have not legitimised the diagnosis of tuberculosis by itself; but, after excluding all other candidate causes of general infection, this data does play a supportive role in strengthening the diagnosis of tuberculosis (however the statistics is not used as a basis for a Bayesian computation, given that this method was not known to clinicians at that time). Yet, this statistical data points generically to tuberculosis, but cannot give any further information about the specific type of tuberculosis. This information is provided by radiological report, by the absence of signs of specific infection and by the presence of the "seeds" on the eye's cornea.

It is important at this point, to mention that Frugoni takes the opportunity to explain his students the different symptoms associated to the three main types of tuberculosis. This is done through etiological considerations. Tuberculosis may, in fact, manifest itself in different forms depending on the way the caseation disseminates in the body. In the pulmonary type, caseation, enters the heart from the right side, and then from there, in the lungs through the pulmonary circulation and produces lung embolization and asphyxia. Instead, if the caseation goes to the left side of the heart, then it enters from there, to the entire body through the systemic circulation and produces a general infection: in this case, you can either have the miliary form (the case under consideration), or the meningeal form, which produces symptoms similar to that of a primary meningitis: headache, neck stiffness, vomiting.

From an epistemological point of view, one is struck by the heterogeneity of the logical methods used all along the way to the final diagnosis. The single steps are generally performed deductively, but these are framed in an abductive framework and are supported by probabilistic considerations. Does this mean that the procedure is incoherent or flawed?

My answer to this question is yes, from a pure epistemological point of view (with some *caveat*), but no from a pragmatic point of view.

Epistemical point of view

The difference between a hypothetical-deductive method and a Bayesian one, is that, in the former, hypotheses are produced along the way and changed through accumulation of negative evidence against the old ones, whilst, instead, in a Bayesian procedure, candidate hypotheses are decided from the beginning, and the accumulation of evidence, should help increase the probability of one to the detriment of the others with no possibility to introduce a new hypothesis on the way. In fact, the Bayesian procedure can be represented as a cake-diagram, where, the growth of a sector necessarily leads to decrease of the area of at least one of the others. In order for this procedure to function, the contemplated hypotheses cannot change during the inferential process (at best they can be eliminated). If new hypotheses need to be added, instead, the old inference process is interrupted and a new one starts, with a new sample space. However, the main diverging point is in the underlying assumption at the basis of the two inferential procedures. In the Bayesian paradigm it is assumed that the probability of the disjunction equals 1: $P(H_1 \vee H_2 \vee \ldots \vee H_n) = 1$. This equates to saying that at least one of the hypotheses must be true. Instead, the hypothetical-deductive paradigm, does not commit to the truthfulness of the hypotheses that pass the elimination test; it just says that they have not been falsified. Therefore, when the set of hypotheses that have not been falsified in a hypothetical-deductive procedure are taken as the starting point for a Bayesian inference, one fails to remember that their disjunction is not guaranteed to be true, and therefore, its probability is not 1. This has both logical and epistemological bearings. From a logical point of view, if the probability assigned to the sample space does not amount to 1, then no computation, at least in the Bayesian method, is possible. From an epistemological point of view, the Bayesian method aims at reducing uncertainty as to which one of the contemplated hypotheses it is the *true* one, whereas, the hypothetical-deductive methods aims at reducing the set of possible worlds by progressively eliminating the false ones, without committing to the truth of the remaining ones.

A possible converging point between the two systems, could be represented by considering the catch-all hypothesis as a translation (in probabilized version) of the *modus tollens* used in the hypothetical-deductive method.

The catch-all hypothesis is simply the partition cell "other". In a sample space of two hypotheses, this would be $\neg H$; it would be $\neg (H_i \vee H_j)$ when the cells are three, and so on. The catch-all hypothesis explicitly accounts for the proportion of ignorance which surrounds the inference. Still, the epistemological assumption of the Bayesian paradigm is that accumulation of evidence should reduce uncertainty and approach truth, which is paradoxically compatible with an increase of *conscious* "ignorance" [e.g. : : $P(H/E_1) > P(\neg H/E_1)$ but $P(\neg H/E_1, E_2, \ldots, E_n)$ may be higher than : $P(H/E_1, E_2, \ldots, E_n)$].[4] Instead, the converse does not hold for the hypothetical-deductive paradigm, which has no instrument for measuring how much falsehood has been eliminated to the benefit of how much truth. On the other hand, the point of the hypothetical-deductive method is not to measure falsehood against truth, but to detect falsehood and then trigger a new abductive process with the hope that the resulting hypotheses pass subsequent tests. Therefore, even if the catch-all hypothesis can be seen as a locus of intersection between the Bayesian and the hypothetical-deductive paradigm, nevertheless it serves heterogeneous purposes in each of them.

Pragmatical point of view

Let's look back at the case history. We could compare the first part of the diagnosis (1 -3) to a simple form of abduction II, where lack of evidence for a specific infection and presence of fever, requires an explanation which is found in the hypothesis of general infection.

[4] But see Stanford (2009) for a debate on this issue.

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From this moment onward, this hypothesis is taken for granted, and the inferential work is devoted to understanding what kind of general infection is at work, rather than testing this hypothesis. The second part of the diagnosis (4-6), is devoted to testing the diverse possible candidates for a diagnosis of general infection. The implications for each of them are investigated and used to eliminate the related hypothesis when failing to be present. In the final part (7-8), the remaining hypotheses are considered as the two possible cells of a sample space and then associated with a probabilistic estimation on the basis of relevant positive and negative evidence.

The hypothetical-deductive and the Bayesian framework are used one after the other by tacitly assuming that the set of non-falsified hypothesis can represent a disjunction whose probability-sum amounts to one. This is done by contradicting the hypothetical-deductive paradigm in two ways: 1) by fixing at some point the hypotheses under consideration, thereby excluding any other hypotheses which might be conceived on a second thought; 2) by considering that at least one of them must be true, whereas, in the hypothetical-deductive framework no truth-commitment is made as to the non-falsified hypothesis.

However, this compromise is useful for many reasons. The eliminative procedure strongly simplifies a process which quickly eliminates candidate hypotheses one after the other; the subsequent use of the probabilistic method allows to arrange the remaining hypotheses in a ranking which is supposed to reflect closeness to truth.

Both inferential processes are important to medical diagnosis: the hypothetical-deductive one is open and flexible, but requires conclusive evidence; the Bayesian method works with simply supporting or disconfirming evidence, but requires a closed set of hypotheses.

Although it is hard to think of a way to reconcile them on a theoretical level, the medical practice constantly does this in the attempt to maximize the information that can be provided by different types of evidence.

Medical diagnosis, especially differential diagnosis, also uses a kind of "probabilistic *modus tollens*", in that, signs (symptoms or laboratory data) are taken as strong evidence for a given hypothesis *not* to be true: the focus is not on hypothesis confirmation, but instead on its refutation [$Pr(\neg H/E_1, E_2, \ldots, E_n)$]. Especially at the beginning of a complicated case, odds are between the hypothesis that is potentially being excluded and a vague "other". This procedure has the advantage of providing a clue for what evidence to look for, and to eventually reduce the set of candidate hypotheses if conclusive negative evidence is found. Indeed, the key issue in the diagnostic procedure is not only to assess the probability of the considered hypotheses on the available evidence, but also how to decide what information to look for.

Conclusion and outlook

Federspil and Vettor (2000) provide a different explanation for the use of the different types of inferential methods in the clinical case presented above (which they also analyse). They defend the position, according to which, not only ethics and rights are governed by dialectic argumentation, rather than pure logic, but also natural science and the hard sciences in general are ruled by rhetorical games and dynamics which are typical of the persuasive discourse. They assert that this is also valid for medicine and clinical decisions, even if the medical profession has generally tended to hide controversies and to present only the logically incontrovertible facts about medical diagnosis. However, in order to demonstrate their thesis, they do not draw on the Frugoni's case, but on another one, which is precisely a court case, where the medical topic hides the shift from a pure clinical domain to a legal one. The case presented is therefore, not adequate for showing anything relevant about pure clinical investigation.

The case they present concerns a young worker who died because of a sudden pulmonary infection some days after his supervisor hit him with a stick on the stomach and on the back. The main question is whether his superior's aggression can be considered the cause of his death.

Three, more or less jointly contributing causes are considered: the physical aggression, the persistent cold that the young man had been exposed to in the last days, and the moral depression caused by the supervisor's hostility. Murri's work, an important clinician in the Italian's medicine history, consists in showing that cold alone could not have by itself caused the pulmonary infection; instead, the sensation of cold, especially perceived after the assault, was itself a consequence of the physical and moral aggression, and together with these other two circumstances, it constituted a complex causal framework, where the triggering phenomenon was to be identified in the physical and moral violence suffered by the victim. Federspil and Vettor's analysis, examines the rhetorical means used in order to persuade the court of his thesis (for instance the argumentum ad verecundiam) and the various logical fallacies made along the way (e.g. petitio principii); however their analysis fails to recognize that the legal framework differs from the clinical one, not only because of the different epistemological and practical purposes and working methods, but also, because generally, the issue is not primarily the diagnosis which, for instance, can be established through autopsy, but the proximate cause for the health injury, because generally, the purpose is to establish the responsibility and its legal liability.

It is however important to emphasise athat the success of the Bayesian paradigm in medical decision-making, is also due to its practical usefulness in case of litigation for the attribution of damage's responsibility in case of medical treatment. In the case above, for instance, a Bayesian computation, made with the help of historical statistics and other available evidence, would have helped the court to assess the single contribution of each causal factor to the final event. This, in turn, would have led to a precise estimation of the complex role played by the assault in causing the young man's death, perhaps leading to a more moderate use of rhetorical instruments.

References

1. Bermudez HL. Decision theory and rationality. OUP, Oxford 2009.
2. Blois MS. Information and medicine. The nature of medical descriptions. University of California Press, Berkley 1984.
3. Bovens L, Hartmann S. Bayesian epistemology. Clarendon Press, Oxford 2003.
4. Burnum JF. Medical diagnosis through semiotics. Giving meaning to the sign. Ann. Intern. Med. 1993 Nov 1;119(9):939-43.
5. Cartwright N. Hunting causes and using them. CUP, Cambridge 2007.
6. Cowell RG, Dawid AP, Lauritzen SL, Spiegelhalter DJ. Probabilistic networks and expert systems. Exact computational methods for Bayesian networks. Springer, New York 2007.
7. Federspil G, Vettor R. La diagnosi clinica tra logica e retorica. In: Galli G. (ed.) Interpretazione e Diagnosi. Scienze umane e medicina. Macerata: Atti del XX Colloquio sulla Interpretazione, 2000.
8. Fox J. Arguing about the evidence: a logical approach. In Dawid P, Twining W, Vasilaki M. (Eds.) Evidence, inference and enquiry. The British Academy, London. In press
9. Fox J. Probability, logic and the cognitive foundations of rational belief. J. Appl. Logic 2003; 1(3-4): 197-224.
10. Fox J, Das S. Safe and sound: artificial intelligence in hazardous applications. The MIT Press, Cambridge 2000.
11. Frugoni C. Lezioni di clinica medica. Padova, 1948
12. Glymour C. What is right with 'Bayes net methods' and what is wrong with 'Hunting causes and using them'? Brit. J. Phi. Sci. Advance Access published on November 12, 2009; doi: 10.1093/bjps/axp039
13. Hacking I. An introduction to probability and inductive logic. CUP, Cambridge 2001.
14. Huemer M. Explanationist aid for the theory of inductive logic. Brit. J. Phi. Sci. 2009; 60(2): 345-375.
15. Kassirer JP. Diagnostic reasoning. Ann. Intern. Med. 1989 Jun 1;110(11):893-900.
16. Kassirer JP, Kopelman RI. Learning clinical reasoning. Williams & Wilkins, Baltimore 1991.
17. Lipton P. Inference to the best explanation. 2nd Ed. Routledge, London 2004.
18. Maddalena G. (a cura di) Peirce. Scritti scelti. UTET, Torino 2005 .
19. McGrew T. Confirmation, heuristics, and explanatory reasoning. Brit. J. Phi. Sci. 2003; 54(4): 553-567.
20. Mushlin S B, Greene HR. Decision making in medicine. An algorithmic approach. Mosby-Elsevier, Philadelphia, Pa 2010.
21. Panzer RJ, Black ER, Griner PF. (Eds.) Diagnostic strategies for common medical problems. American College of Physicians, Philadelphia, Pa 1991.
22. Peirce CS. Lecture Two: types of reasoning. In Ketner KL. ed.; Peirce CS. Reasoning and the logic of things, The Cambridge conference lectures of 1898. Harvard University Press, Cambridge (etc.) 1992: 123-142.
23. Peirce CS. Harvard lecture 1, Spring 1865 (MS 94). In Moore EC, Fisch MH. et al. (eds.) Writings of Charles S. Peirce. Volume I (1857–1866). Indiana University Press, Bloomington, Indiana 1981.
24. Hartshorne C, Weiss P. (Eds.); Peirce CS. Collected papers. Belknap Press of Harvard University Press, Cambridge, Mass. 1934.
25. Peterson M. An introduction to decision theory. CUP, Cambridge 2009.
26. Petöfi JS. La diagnosi come semiotica applicata. Osservazioni sulla storia della semiotica e sulla concezione di Peirce. In Galli G. (ed.) Interpretazione e diagnosi. Scienze umane e medicina. Macerata: Atti del XX Colloquio sulla Interpretazione, 2000.
27. Popper K. The logic of scientific discovery. Routledge, London 2002.
28. Psillos S. Inference to the best explanation and Bayesianism. Institute of Vienna Circle Yearbook 11. Kluwer, Dordrecht 2004.
29. Salmon WC. Explanation and confirmation: A Bayesian critique of inference to the best explanation. In Hon G, Rakover SS. (Eds.) Explanation: theoretical approaches and applications. Kluwer Academic Publishers, Dordrecht (etc.) 2001.
30. Salmon WC. Reflections of a bashful Bayesian: A reply to Peter Lipton. In Hon G, Rakover SS. (Eds.) Explanation: theoretical approaches and applications. Kluwer, Dordrecht (etc.) 2001.
31. Stanford PK. Scientific realism, the atomic catch-all hypothesis: Can we test fundamental theories against all serious alternatives Advance Access published online on February 19, 2009 ;doi:10.1093/bjps/axp003
32. Vitalcolonna L. Principi e contributi di semiotica del testo. Bulzoni, Roma 1999.
33. Weisberg J. Locating IBE in the Bayesian framework. Synthese 2009; 167(1): 125-143.
34. Thagard P. Computational philosophy of science. MIT Press/Bradford Books, Cambridge, Mass 1988.